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Part II

Environmental Protection Agency

Clean Water Act, Toxic Pollutant List; Removal of Dichlorodifluoromethane and Trichlorofluoromethane; Petition to Remove Ethylbenzene, Phenol, 2,4-Dichlorophenol, 2,4,5-Trichlorophenol, and Pentachlorophenol; Proposed Denial of Petition to Remove Monochlorophenyl Phenyl Ether; and Petition to Remove Chlorodifluoromethane

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 401

[WH-FRL 1701-5]

Removal of Dichlorodifluoromethane and Trichlorofluoromethane From the Toxic Pollutant List Under Section 307(a)(1) of the Clean Water Act

AGENCY: United States Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: EPA is deleting dichlorodifluoromethane (F–12) and trichlorofluoromethane (F–11) from the toxic pollutant list. As a result of its review of available data and the public comments, the Agency concludes that no significant potential exists for exposure to the two chemicals via water. This conclusion coupled with the compounds' low mammalian toxicity does not suggest any environmental or human health hazard resulting from the delisting of these compounds. EPA has determined that deleting these compounds will not compromise adequate control over their discharge into the aquatic environment and EPA anticipates no adverse effects on the aquatic environment as a result of this action.

EFFECTIVE DATE: December 24, 1980.

FOR FURTHER INFORMATION CONTACT: Jacqueline V. Carr, Criteria and Standards Division (WH–585), Office of Water Regulations and Standards, U.S. Environmental Protection Agency, 401 M Street SW, Washington, D.C. 20460 (202–245–3036).

SUPPLEMENTARY INFORMATION:

Background

On November 2, 1979, E. I. du Pont de Nemours and Company petitioned EPA to remove the fluorocarbons, dichlorodifluoromethane (F–12) and trichlorofluoromethane (F–11) from the toxic pollutant list established under section 307(a)(1) of the Clean Water Act. After reviewing data supplied by Du Pont to support the petition and additional data, EPA published a proposal to delist F–11 and F–12 and requested comment on the proposal in 45 FR 46103 July 9, 1980.

Four respondents submitted comments. All respondents supported the action to remove the chemicals from the toxic pollutant list.

As a result of its review, EPA concludes that available data support the deletion of dichlorodifluoromethane (F–12) and trichlorofluoromethane (F–11) for the following reasons:

(1) No significant potential exists for contamination of water supplies by F–11 or F–12 due to the methods of transportation, use, and disposal of these compounds. During manufacture of these compounds, total losses to the environment are small. Almost all of the total losses during manufacturing are to the air from fugitive emissions with only a miniscule amount of loss accounted for by direct discharge into water from point source effluents.

(2) Experimental data and field measurements all confirm the water/air partition for F–11 and F–12 is so low that no significant hazard exists for contamination of water supplies.

(3) The low solubility and high volatility of F–11 and F–12, combined with low mammalian toxicity do not suggest any environmental or human health hazard.

Dated: December 24, 1980.

Douglas M. Costle,

Administrator.

40 CFR Subchapter N, Part 401 is amended by the deletion of two compounds.

§ 401.15 [Amended]

At § 401.15 under number 38, halomethanes, in the list of toxic pollutants designated pursuant to section 307(a)(1) of the Act, delete both trichlorofluoromethane (F–11) and dichlorodifluoromethane (F–12).

[FR Doc. 81–175 Filed 1–7–81; 8:45 am]

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ENVIRONMENTAL PROTECTION AGENCY

[WH-FRL 1701-6]

Petition To Remove Ethylbenzene, Phenol, 2,4-Dichlorophenol, 2,4,5-Trichlorophenol, and Pentachlorophenol From the § 307(a)(1) List of Toxic Pollutants Final Action

AGENCY: United States Environmental Protection Agency.

ACTION: Denial of a petition from the Dow Chemical Company to remove ethylbenzene, phenol, 2,4-dichlorophenol, 2,4,5-trichlorophenol, and pentachlorophenol from the list of toxic pollutants under section 307(a)(1) of the Clean Water Act, as amended, 33 U.S.C. 1317(a).

SUMMARY: On November 7, 1979, in 44 FR 64555, the EPA published and requested public comment on a petition and supporting data from Dow Chemical Company, USA to remove five chemicals from the section 307(a)(1) toxic pollutant list. Ten respondents submitted comments to the Agency.

EPA reviewed all supporting data supplied by Dow, the public comments, and additional available data and concludes that the present data base supports the continued listing of phenol, 2,4-dichlorophenol, 2,4,5-trichlorophenol,and pentachlorophenol based on their mammalian and aquatic toxicity, widespread occurrence in the aquatic ecosystem and potential to persist under certain aquatic conditions. Available data also support the continued listing of ethylbenzene because it occurs in the aquatic environment, bioacumulates in man, and has the potential to persist in the aquatic ecosystem.

Discharge of chemicals listed under section 307(a) of the Clean Air Act are subject to the most stringent technological controls representing Best Available Technologies Economically Achievable (BAT). BAT is anticipated to be more costly than technologies resulting from waivers from BAT (granted to industrial dischargers of nonconventional pollutants) or technology for conventional pollutants. The denial of this petition precludes reclassification of the chemicals thereby eliminating the possibility for industrial use of alternative technologies and resultant lessening of economic impact.

FOR FURTHER INFORMATION CONTACT: Jacqueline V. Carr, Criteria and Standards Division (WH-585), Office of Water Planning and Standards, U.S. Environmental Protection Agency, 401 M St., SW., Washington, D.C. 20460 (202-245-3036).

SUPPLEMENTARY INFORMATION:

Background

Pursuant to section 307(a) of the Clean Water Act of 1977 (the Act), EPA published a list of toxic pollutants on January 31, 1978 in 43 FR 4109. Section 307 also authorizes the Administrator to add or remove any pollutant from the list. In revising the toxic pollutant list, the Administrator is directed to take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of affected organisms in any waters, the importance of the affected organisms, and the nature and extent of the effect of the pollutant on such organisms.

Listing determinations under Section 337(a) are discretionary. The Act lists a number of factors the Administrator "shall take into account", but allows a weighing of these factors, leaving ample leeway for discretionary action.

On August 11, 1978, Dow Chemical Company, U.S.A. (Dow) requested that ethylbenzene, phenol, 2,4-dichlorophenol, 2,4,5-trichlorophenol, and pentachlorophenol be removed from the toxic pollutant list. Dow claimed that the five chemicals fail to meet the statutory criteria for toxic pollutants under section 307(a)(1). Dow asserts specifically that:

(1) Ethylbenzene, phenol, 2,4,5-trichlorophenol and pentachlorophenol are not persistent in the environment. Removal mechanisms include biodegradation and/or volatilization.

(2) No evidence has been cited for the carcinogenicity, teratogenicity, or mutagenicity of ethylbenzene, 2,4-dichlorophenol, 2,4,5-trichlorophenol and pentachlorophenol, and phenol.

(3) Toxicological findings indicate that ethylbenzene is only moderately toxic to fish and that 2,4-dichlorophenol, and 2,4,5-trichlorophenol have low mammalian toxicities via oral exposure.

Dow also asserted that retention of these five chemicals on the list dilutes the Agency's efforts and diverts attention from more serious environmental issues; introduces unwarranted barriers to the introduction of new products containing the compounds, often resulting in the use of less efficacious or more expensive materials; and results in the needless expenditure of funds by industry and government for expensive monitoring, analysis and reporting.

On November 7, 1979, in 44 FR 6455, the EPA requested public comment on Dow's petition. Ten respondents submitted comments. Three respondents supported the continued listing of ethylbenzene, phenol, 2,4-dichlorophenol, 2,4,5-trichlorophenol and pentachlorophenol as toxic pollutants. One respondent supplied information without any recommendation. The remaining respondents supported specific delistings: three supported the delisting of phenol; two supported the delisting of pentachlorophenol; and one supported the delisting of phenol and ethylbenzene.

Action on the Petition

The EPA concludes that the petition from Dow to delist phenol, 2,4-dichlorophenol, 2,4,5-trichlorophenol, pentachlorophenol and ethylbenzene must be denied because available data indicate that:

(1) Phenol warrants listing because of the combined effects of its high toxicity to aquatic life, its ability to promote the carcinogenicity of weak carcinogens, its widespread occurrence in industrial effluents and the aquatic ecosystem, and its potential to persist under certain environmental conditions.

(2) Pentachlorophenol warrants listing because it is highly toxic to aquatic organisms, bioaccumulates, and is widespread in industrial effluents and in the aquatic ecosystems.

(3) 2,4-Dichlorophenol and 2,4,5-trichlorophenol warrant listing because they are highly toxic to aquatic organisms, they promote the carcinogenicity of other chemicals and they are discharged in point source effluents.

(4) Ethylbenzene warrants listing because it occurs in industrial effluents and the aquatic environment and it bioaccumulates in man any may bind to sediment offering a potential for exposure to benthic organisms. The current toxicity data base for ethylbenzene is insufficient to warrant delisting since there is no assurance that adequate control of this chemical will not be compromised (Cong. Reg. daily ad. S. 19649).

Summary of Aquatic Fate

Persistence/Degradability/Occurrence

The biochemical oxygen demand (BOD) data which Dow supplied as evidence that the chemicals do not persist due to rapid biodegradation were theoretical calculations based on laboratory tests performed under experimental conditions in which oxygen, nutrients and acclimated organisms were in abundance. This situation rarely exists in nature. Furthermore, there is evidence which suggests that the theoretical degradation

rates reported by Dow are significantly higher than those actually observed in nature. Data relevant to each particular compound are summarized below.

Phenol

Dow asserted that phenol is more than 99 percent biodegraded in seven days. However, a report of a 1974 derailment in southern Wisconsin which resulted in significant groundwater contamination by phenol indicated that nineteen months after the spill, phenol still persisted (Delfino and Dube, 1976; Baker, et. al., 1978). The first tests revealed phenol concentrations of 0.21 to 3.2 mg/l in nearby wells. Concentrations in the well water eventually reached a maximum of 1,130 mg/l. These data indicate that the rates of dilution and degradation were very slow, and depends, in part, on the amount of the chemical in the environment and other physico-chemical conditions.

Phenol is highly soluble in water, indicating that exposure to affected organisms is quite likely. The water solubility of phenol ranges from 67,000 mg/l at 16°C to essentially complete miscibility at 66°C (Kirk and Othmer, 1963), suggesting a potential for exposure in excess of acutely toxic levels. It has been detected (but not quantified) in the wastewaters of various industries, in finished drinking water, sediment, and fish (Shackelford and Keith, 1976; U.S. EPA, 1980).

Large amount of the chemical are produced annually. Phenol ranked 38th in production among U.S. chemicals in 1978 (*Chemical & Engineering News*, 1979) with annual production of 2.38 billion pounds (United States International Trade Commission, 1978), so that the likelihood of discharge to the aquatic environment is high. Disposal methods include deepweel injection, incineration, degradation in water and municipal sanitation (NSF, 1975). Deepweel injection is another potential source of contamination of groundwater, and (eventually) of surface water.

2,4-Dichlorophenol and 2,4,5-Trichlorophenol

Few data exist regarding the persistence of chlorinated phenols in the environment. Although microbial biodegradation has been extensively studied, data on other environmental processes are limited. Dow concluded that 2,4-dichlorophenol and 2,4,5-trichlorophenol were rapidly degraded and therefore nonpersistent in the aquatic environment. Dow based its conclusion on theoretical oxygen demand data which showed 2,4-dichlorophenol to be 90 percent degraded at five days with no further degradation by 20 days and 2,4,5-trichlorophenol to be 44 percent degraded at 5 days and only 75 percent degraded at 20 days. Dow's conclusion that 2,4,5-trichlorophenol is rapidly biograded is inconsistent with their definition of rapidly biodegradable chemicals, i.e. those chemicals which are at least 50 percent biodegraded in 5 days. In fact, at 20 days, 25 percent of the 2,4,5-trichlorophenol persisted in Dow sample. Alexander and Aleem (1961) reported that 2,4,5-trichlorophenol was resistant to microbial decomposition. Additional data indicate that 2,4-dichlorophenol is biodegraded but that the rates of microbial degradation are dependent on numerous variables, so that the compound can be expected to persist under certain conditions. The low vapor pressure (1 mm Hg at 53.0°C) and non-volatility of 2,4-dichlorophenol from alkaline solutions (Sax, 1975) would cause it to be only slowly removed from surface water via volatilization. Studies have indicated low sorption of 2,4-dichlorophenol from natural surface waters by various clays (Aly and Faust, 1964). Aly and Faust (1964) examined the dissipation of 2,4-DCP from natural lake waters at a buffered pH of 7. In aerated lake waters, with initial 2,4-DCP concentrations of 100, 500, and 1,000 ug/l, the percentages of 2,4-DCP remaining at 9 days were 0, 0.34, and 46 respectively. By contrast, initial concentrations of 100, 500, and 1,000 ug/l in unaerated and unbuffered waters resulted in percentages of 40, 51.6, and 56, respectively, remaining at 17 days. Aly and Faust concluded that the persistence of chlorophenol would tend to increase at lower pH and under anaerobic conditions that might result from the decomposition of excessive organic matter.

Ingols, et al. (1966) studies the degradation of various chlorophenols by activated sewage sludge and concluded that 2,4-DCP was degraded more rapidly by activated systems with previous exposure to chlorophenols than by by those with no previous exposure to chlorophenols. When activated sludge was exposed to 2,4-DCP at levels of 100 mg/l of sludge, 75 percent of the chemical disappeared in two days, and essentially 100 percent was gone in five days. However, acclimated organisms are not expected to be found in nature.

2,4-dichlorophenol has been detected in municipal and industrial effluents, finished drinking water, and surface waters (Shackelford and Keith, 1976).

Sidewell (1971) verified the presence of 2,4-dichlorophenol in 2,4-dichlorophenoxyacetic acid manufacturing wastes. Over a 3-month period, total chlorophenol content ranged from 68 mg/l of waste to 125 mg/l, with 2,4-dichlorophenol content ranging as high as 86 percent of total.

Trichlorophenol (unspecified isomers) has been found in river water, finished drinking water, chemical plant effluent water and sewage treatment plant effluents in the U.S. (Shackelford & Keith, 1976) indicating environmental persistence. 2,4,5-Trichlorophenol was detected at unspecified concentrations in drinking water (Deinzer et. al., 1975). 2,4,5-Trichlorophenol was found at levels of 16–45 mg/kg in body fat of rainbow trout after experimental exposure to sulphate pulp bleachery effluents diluted 40 times with brackish water (Landner et. al., 1977). No measured bioconcentration factor is available for 2,4,5-trichlorophenol, but data on octanol/water partition coefficients indicate that 2,4,5-trichlorophenol should bioaccumulate to a greater extent than 2,4,5-trichlorophenol, thereby increasing the potential for exposure to consumers of aquatic life. Partition coefficients of 6,000 and 4,900 were reported for 2,4,5- and 2,4,5-trichlorophenol, respectively (U.S. EPA, 1979c).

Pentachlorophenol

Dow asserts that a number of studies indicate that pentachlorophenol does not persist in the environment. Dow reported that Arsenault (1976) summarizes naturally occurring detoxification mechanisms, i.e., biodegradation, degradation by light, methylation, and conjugation with sulfate (detoxified bound form). Dow also stated that Chue and Kirsch (1972) and Etzel and Kirsch (1975) demonstrated the biodegradability of pentachlorophenol in continuous-flow enrichment cultures. Pentachlorophenol concentrations as high as 200 ppm were metabolized as the sole source of organic carbon and energy, the product of metabolism being carbon dioxide (CO_2).

Data available to EPA indicate that pentachlorophenol is persistent in water and in aquatic organisms. Pentachlorophenol has been detected in the effluent waters from various manufacturing and processing plants (Shackelford & Keith, 1976), in rain-, snow- and lake-water, and in creek-water containing industrial discharges (Fountaine et al., 1976). Pentachlorophenol also has been detected in fish, white shark liver, bird eggs anf fish food (Zitko et al., 1974), birds, snails, frogs and fish (Vermeer et

al., 1974; Renberg, 1974), and fish extracts (Tokunaga, 1971).

The log octanol/water partition coefficient of 5.01 (Leo et al., 1971) indicates that pentachlorophenol should be bioaccumulated significantly in the aquatic environment. Several investigations have documented the distribution of pentachlorophenol in the aquatic environment, confirming its potential to bioaccumulate. Rudling (1970) observed a 1,000-fold concentration of the compound in the eel, *Anguilla anguilla,* living in lake water that had been contaminated with a 3 μg/l concentation of pentachlorophenol from pulp mill discharges. Pierce and Victor (1978) studied the fate of pentachlorophenol in a freshwater lake near Hattiesburg, Mississippi, after accidental spills of wood-treating pentachlorophenol-containing wastes in fuel oil. Pentachlorophenol was found to persist in the water and in fish over six months following the spills. Fish were also observed to accumulate several of the degradation products of pentachlorophenol, namely pentachloroanisole and the 2,3,5,6- and 2,3,4,5-tetrachlorophenol isomers. The bioaccumulation of pentachloroanisole is to be expected, based upon its log octanol/water partition coefficient of 5.66 (Tute 1971).

Akitake and Kobayashi (1971) demonstrated that a 72-hour exposure to a sublethal level of pentachlorophenol of 100 μg/l resulted in a 900-fold concentration of the compound in the goldfish, *Carassius auratus.* In another study (Kobayashi and Akitake, 1975) these authors reported a concentration factor of 1,000 after exposure of goldfish for 120 hr. in 0.1 mg/l pentachlorophenol and observed a maximum concentration of 166 μg pentachlorophenol/g body weight in fish exposed to 0.2 mg/l in the water. Lee and Metcalf (1975) studied the fate of radiolabeled pentachlorophenol in a model aquatic ecosystem with a six element food chain and reported it to be ecologically magnified (bioconcentrated) in the mosquito fish, *Gambusia affinis.*

Ethylbenzene

Its vapor pressure and water solubility suggest that volatilization is one mechanism for removal of ethylbenzene from the aquatic environment (U.S. EPA, 1979b; Dow, unpublished). The relative importance of biodegradation in the determination of the fate of ethylbenzene in the aquatic environment is not clear (U.S. EPA, 1979b). Dow's data suggest that only forty-five percent of their ethylbenzene sample was biodegraded after 20 days (Dow, unpublished). Although no specific environmental sorption studies were found in the reviewed literature, the log octanol/water partition coefficient of 3.15 (Tute 1971) suggests that sorption processes may be significant for ethylbenzene. The extent to which this adsorption will interfere with volatilization has not been determined.

Ethylbenzene has been detected at several geographical locations in finished drinking water, industrial effluents, surface waters and well waters (Burnham, et al., 1972; Shackelford and Keith 1976). In a survey of water contamination present in the drinking water of ten cities in the United States, ethylbenzene was detected but not quantified in six of ten samples (U.S. EPA, 1975). Recent studies of the coastal waters of the Gulf of Mexico have shown that aromatic hydrocarbons comprise 80–90% of the total dissolved hydrocarbons at most sampling sites. Total concentrations of volatile aromatic such as ethylbenzene range from 0.6 to 4.5 ng/liter (Sauer, 1978).

Ethylbenzene has been shown to persist in man for days after exposure (Wolff, et al. 1977). It is present in the respiratory tract (Conkle, et al. 1975), umbilical cord and maternal blood (Dowty, et al. 1976) and subcutaneous fat (Wolff, et al. 1977) of exposed humans.

Summary of Toxicological Effects

Phenol

Aquatic Life Toxicology

Toxicity data for phenol indicate clearly that the compound is sufficiently toxic to justify continued listing under section 307(a). An EPA report cites acute toxicity data for phenol in 13 species of freshwater fish and 15 species of freshwater invertebrates (U.S. EPA, 1979a). All of these organisms are important to the aquatic food chain and most reside in U.S. waters. Rainbow trout, the fish species most sensitive to phenol, had LC 50 concentrations as low as 5 mg/l (McLeary, 1976). These results are confirmed by data submitted by Dow from a study in its laboratories which listed an LC 50 for phenol in rainbow trout of 4.97 mg/l (Brosier, 1974). Phenol was fatal to rainbow trout at 7.3 mg/l in 2 hours and at 6.5 mg/l in 12 hours; at these concentrations there was rapid damage to gills and severe pathology of other tissues (Mitrovic, et al. 1968). Pathologic changes in gills and in fish tissues were found at phenol concentrations in the range of 20 to 70 μg/l (Reichenbach-Klinke, 1965).

The one available acute LC 50 value for phenol in marine fish was 6.0 mg/l in the mountain bass, *Kuhlia sandvicensis,* (Nunogowa et al., 1970). Acute LC 50 values for phenol in marine invertebrates were 58 mg/l in the eastern oyster, *Crassostrea virginica* and 52 mg/l in the hard clam, *Mercenaria mercenaria.* Decreased egg development in the oyster, *Crassostrea virginica,* has been found to occur at phenol levels of 2 mg/l (Davis and Hidu, 1969). Various environmental conditions will increase the toxicity of phenol. Lower dissolved oxygen concentrations, increased salinity, and increased temperature ali enhance the toxicity of phenol (EIFAC, 1973).

Mammalian and Human Toxicology

Regardless of the route of administration of phenol, the signs and/or symptoms of acute toxicity in man and experimental animals are similar. The predominant acute action of a toxic dose of phenol in man appears to be on the central nervous system, leading to sudden collapse and unconsciousness. In some mammalian species, these effects are preceded by muscular twitchings and severe convulsions. The approximate lethal doses (LD_{50}) for phenol in various species exposed by oral and dermal routes include oral doses of 0.1 g/kg, 0.5 g/kg, 0.4 to 0.6 g/kg and 0.34 to 0.53 g/kg in the cat, dog, rabbit and rat, respectively. The LD_{50} of phenol in rats via the dermal route of exposure was 0.67 to 2.5 g/kg (U.S. EPA, 1980). It is difficult to estimate the LD_{50} for oral exposure to phenol for man, even though phenol has a long history of use in suicidal attempts. Oral doses of phenol estimated to be lethal to man range from 0.14 g/kg to 0.43 g/kg (U.S. EPA, 1980b).

The symptoms reported by humans who had consumed phenol-contaminated groundwater for approximately one month (Baker, et. al. 1978) included diarrhea, mouth sores and burning mouth. The daily dose of phenol consumed was estimated to be 10 to 240 mg.

Heller and Pursell (1938) fed phenol to rats in their drinking water concentrations ranging from 100 to 12,000 mg/l over several generations. Impairment of growth was observed at concentrations between 7,000 and 12,000 mg/l.

In an unpublished study by Dow Chemical Company (1976), rats were fed by gavage 20 daily doses of 0.1 g phenol/kg body weight. These rats showed slight liver and kidney effects, while rats which received 20 daily doses of 0.05 or 0.01 g phenol/kg body weight demonstrated none of those effects. In a subsequent series of tests, rats received 135 doses of 0.1 or 0.05 g phenol/kg body

weight by gavage over a 6-month period. The growth of the rats receiving the phenol was comparable to that of the controls. Very slight liver changes and slight to moderate kidney damage were seen in the rats which had received 0.1 g phenol/kg. The feeding of 0.05 g phenol/kg resulted only in slight kidney damage.

In a 41-day feeding study, Kociba, et al. (1976) fed 125 mg phenylsalicylate/kg/day to beagle dogs. Since phenylsalicylate is metabolized to phenol, this resulted in urinary levels up to 6,144 mg/l. This high level of phenol excretion was not associated with any discernible ill effects in the dogs. Repeated exposures to phenol at high concentrations have resulted in chronic liver damage in man (Merliss, 1972).

Phenol has been reported to be mutagenic (without metabolic activation) *vitro* to *Drosphila* gonadal tissue (Hadorn and Niggli, 1946), and in *Escherichia coli* B/sd-4 at concentrations of 0.01 to 0.2 percent and bacterial survival rates were 0.5 to 1.7 percent, respectively (Demerec et. al., 1951). Phenol was not mutagenic (with or without metabolic activation) in six mutant strains of *Salmonella typhimurium* (Cotruvo et. al., 1970 and *Neurospora* (Dickey et. al., 1949).

Salaman and Glendenning (1957), Boutwell and Bosch (1959), and Van Duuren et. al. (1968) have shown that phenol in acetone or benzene promotes skin cancer in several strains of mice pretreated with dimethylbenz(a)anthracene or benzo(a)pyrene.

2,4-Dichlorophenol, 2,4,5-Trichlorophenol and Pentachlorophenol

Aquatic Life Toxicology

There are few data on the acute toxicity of 2,4-dichlorophenol, 2,4,5-trichlorophenol, and pentachlorophenol and no chronic toxicity data are available for aquatic organisms.

Two studies on the acute toxicity of 2,4-dichlorophenol to freshwater fish are available. Ninety-six hour LC 50 values of 0.38 mg/l, and 2.02 mg/l were reported for the rainbow trout, *Salmo gairdneri* and bluegill *Lepomis macrochirus*, respectively (Brosier 1974; U.S. EPA, 1979d). Two studies on *Daphnia magna* report 48-hour LC 50 values of 2.6 mg/l (Kopperman, et. al. 1974; U.S. EPA, 1978). There are no data available for the acute toxicity of 2,4-dichlorophenol to marine organisms.

Freshwater acute LC 50 values reported for 2,4,5-trichlorophenol are 0.45 mg/l for the bluegill, *Lepomis machrochirus*, 2.7 mg/l for a cladoceran, *Daphnia magna* and EC 50 values of 1.2 mg/l and 1.6 mg/l for the plants *Selenastrum capricornutum* and *Lemna minor*, respectively (U.S. EPA, 1978; Blackman, et. al., 1955). The lowest concentrations of 2,4,5-trichlorophenol reported to kill 50% or more of rainbow trout, *Salmo gairdneri* were 1 mg/l at 48 hours (Shumway and Palensky, 1973) and 0.30 mg/l at 96 hours (Brosier, 1974).

Available saltwater acute LC 50 studies of 2,4,5-trichlorophenol report values of 1.7 mg/l in the sheephead minnow, *Cyprinodon variegatus* and 3.8 mg/l in the mysid shrimp, *Mysidopsis bahia* (U.S. EPA, 1978).

Studies of the acute toxicity of pentachlorophenol to freshwater organisms report LC 50 values of 46 ug/l for rainbow trout, *Salmo gairdneri* (Brosier, 1974) and 63 ug/l for sockeye salmon, *Oncorhynchus nerka* (Webb and Brett, 1973). Sublethal effects were observed on the growth rate of sockeye salmon at a concentration of 1.74 ug/l.

Mammalian and Human Toxicology

Relatively few studies of the acute or subacute toxicity of 2,4-dichlorophenol have been reported. Acute oral LD_{50} values reported are 580–4000 mg/kg in the rat and 1600 mg/kg in the mouse (Deichman, 1943; Kobayaski, et. al. 1972).

The mechanism of toxic action for 2,4-dichlorophenol in mammilian systems *in vivo* has not been well defined. Limited *in vitro* studies indicate 2,4-dichlorophenol inhibits oxidative phosphorylation in rat liver mitochondria and rat brain homogenates (Farquharson, et al. 1958; Mitsuda, et al. 1963). A concentration of 4.2×10^{-5} M 2,4-DCP inhibited oxidative phosphorylation by 50 percent in rat liver mitochondria.

The clinical signs of acute poisoning with 2,4,5-trichlorophenol include decreased activity and motor weakness (Deichman, 1943). Oral LD_{50} values for 2,4,5-trichlorophenol in rats have been reported as 820 mg/kg (in fuel oil) and 2960 mg/kg (in corn oil) (Deichman and McCollister et al., 1961).

Rabbits given 28 daily oral doses of 2,4,5-trichlorophenol in 5 percent gum acacia solution exhibited no effects at 1 or 10 mg/kg but experienced kidney and liver lesions at 100–500 mg/kg (McCollister, et al., 1961).

Rats fed dietery levels of 2,4,5-trichlorophenol of 100, 300, 1000, 3000, or 10,000 mg/kg in feed for 98 days exhibited no effects from ingesting up to 1000 mg/kg. Dietary levels of 3000 and 10,000 mg/kg produced degenerative changes in the kidney and liver, which were considered to be reversible (McCollister et al., 1961). The mechanism of action involves the uncoupling of oxidative phosphorylation (Mitsuda, et al., 1963).

McCollister, et al. (1961) reported on skin irritation and sensitization studies in 200 humans. A 5 percent solution of 2,4,5-trichlorophenol in sesame oil was mildly irritating in a few individuals upon prolonged contact, but there was no evidence of sensitization.

The oral lethal dose of pentachlorophenol in several species of animals ranges from 70 to 300 mg/kg (Bevenue and Beckman, 1967; Diechmann, et al., 1942). The mechanism of action involves the uncoupling of oxidative phosphorylation (Weinbach and Garbus, 1965). Fuel oil-type solvents reduce the lethal dose, while aqueous solutions of the sodium salt are less toxic.

Pentachlorophenol exposure has resulted in death in man through occupational accidental exposures and suicide attempts (Gordon, 1956; Bergner, et al. 1965; Armstrong, et al. 1969). Lesions following fatal exposures include inflamed gastric mucosa, pulmonary congestion, pulmonary edema, fatty metamorphosis of the lever, and degeneration of renal tubules and myocardium.

Symptoms in chronic toxicity, in general, are similar to those seen in acute intoxications. Chronic intoxications result from relatively high levels of continuous exposure. Symptoms in nonfatal chronic exposures include muscle weakness, headache, anorexia, abdominal pain, and weight loss in addition to skin, eye, and respiratory tract irritation.

Pentachlorophenol solutions can cause skin irritation. Immersion of hands for 10 minutes in a 0.4 percent solution of pentachlorophenol cause pain and inflammation (Bevenue, et al. 1967a).

One unique poisoning episode involved babies wearing diapers rinsed in an antimicrobial laundry neutralizer containing sodium pentachlorophenate. Babies wearing the diapers an average of eight days became ill and some died. Some were less severely affected and recovered spontaneously (Armstrong, et al. 1969; Robson, et al. 1969). Six of the nine severely affected had hepatomegaly and two of the nine had splenomegaly in addition to profuse sweating hyperpyrexia.

Goldstein, et al. (1977) fed rats 20, 100, or 500 ppm technical and pure pentachlorophenol (equivalent to 1.2, 6, and 30 mg/kg, respectively) for eight months. At 20 ppm, liver aryl hydrocarbon hydroxylase and glucuronyl transferase were increased in female rats fed technical pentachlorophenol as compared to

controls fed pure pentachlorophenol. At 100 ppm technical pentachlorophenol increased excretion of uroporphyrin and delta-aminolevulinic acid. Feeding 20 or 100 ppm of pure pentachlorophenol had no effect. Body weight gain was reduced at 500 ppm with both types of pentachlorophenol. The no-observable-adverse-effect-level (NOAEL) for pure pentachlorophenol from this study was 6 mg/kg (i.e., the 100 ppm diet group).

Available data do not permit an evaluation of the carcinogenicity of 2,4-dichlorophenol, 2,4,5-trichlorophenol, and pentachlorophenol. The primary carcinogenicity of 2,4-dichlorophenols and 2,4,5-trichlorophenol could not be evaluated from a single study by Boutwell and Bosch (1959) which indicated that 2,4-dichlorophenol and 2,4,5-trichlorophenol promote the tumorigenicity of dimethylbenzanthracene in mice. The report of Boutwell and Bosch (1959) is the only one found that deals with the tumorigenicity of 2,4-dichlorophenol and 2,4,5-trichlorophenol. However, since the study was designed primarily to detect promoting activity, the effects of the compounds as primary carcinogens could not be evaluated (there were no animals treated with 2,4-dichlorophenol or 2,4,5-trichlorophenol alone and length of exposure was 16 weeks rather than full life). The National Cancer Institute (NCI) is scheduling a study of the carcinogenicity of 2,4-dichlorophenol for February 1981. No decision has been made to test 2,4,5-trichlorophenol (NCI, personal communication).

Several studies were found which addressed the mutagenicity of 2,4-dichlorophenol 2,4,5-trichlorophenol and pentachlorophenol. Amer and Ali (1968, 1969) reported some effects of 2,4-dichlorophenol, 2,4,5-trichlorophenol and pentachlorophenol on chromosomes which affected mitosis of *Vicia faba*. Rasanen, et al. (1977) tested 2,4,5-trichlorophenol for mutagenicity using the *Salmonella*-mammalian microsome Ames test with both the nonactivated and activated systems and reported it as nonmutagenic under both conditions. Pentachlorophenol was not mutagenic in *Drosophila melangaster* (Vogel & Chandler, 1974), the mouse host-medicated assay or *in vitro* spot tests (Buselmaier, et al., 1973) and *in vitro* tests with *Salmonella typhimurium* Anderson 35 al., 1972).

No studies are available on the teratogenicity of 2,4-dichlorophenol and 2,4,5-trichlorophenol. Pentachlorophenol has been reported to be nonteratogenic but embryotoxic in rats (Schwetz et al., 1974) and hamsters (Hinkle, 1973). Hinkle (1973) found fetal deaths and/or resorptions in three of six test groups using Golden Syrian hamsters. Dose-response data and statistical analysis were not provided. The dose range was from 1.25 to 20 mg/kg.

Schwetz, et al. (1974) provided more complete data from a rat study using purified and commercial grade pentachlorophenol. Dosages ranged from 5 to 50 mg/kg daily and exposure was during days 6 to 15 of gestation. The NOAEL based on incidence of fetal resorption was 5.8 mg/kg (adjusted dose to provide 5 mg pentachlorophenol/kg) for commercial and 15 mg/kg for purified grade pentachlorophenol. At 50 mg purified pentachlorophenol/kg fetal resorption was 100 percent. The NOAEL level for reducing fetal body weight was 15 mg/kg for both grades. Fetal anomalies consisting of subcutaneous edema and dilated ureters were observed in soft tissues at doses of 15 mg/kg or above for both grades of pentachlorophenol. The NOAEL for soft tissue anomalies was 5 mg commercial grade pentachlorophenol/kg/day. Delayed ossification of the skull was noted at 5 mg/kg with purified pentachlorophenol. The NOAEL for skeletal anomalies with commercial PCP was 5.8 mg/kg. At higher dosages, skeletal anomalies consisted of lumbar spurs, supernumerary or fused ribs, or supernumerary, abnormally shaped, missing, or unfused centers of ossification of vertebrae or sternebrae. These effects were mor readily produced when dosing occurred on days 8–11 rather than days 12–15 of gestation. The authors considered the effects by PCP to be evidence of embryo-toxicity and fetotoxicity, not teratogenicity (U.S. EPA, 1980 b).

Ethylbenzene

Aquatic Life Toxicology

Reported acute LC–50 values for ethylbenzene in freshwater vertebrates and invertebrates were generally between 30 and 200 mg/l (Pickering and Henderson, 1966; U.S. EPA, 1978). One chronic test indicated that effects on the embryo and larval stages of the fathead minnow would occur at concentrations greater than 440 μg/l. No chronic tests have been conducted with saltwater organisms and ethylbenzene and no definite effect level has been determined for freshwater organisms.

Mammalian and Human Health Effects

There have been no investigations of the mutagenicity, carcinogenicity or teratogenicity of ethylbenzene. There are also no recent reports of the chronic toxicity of ethylbenzene from full-time studies in mammals. Wolf et al (1956) reported that ethylbenzene had low acute oral toxicity in rats exposed for 6 months to the chemical. Ethylbenzene caused slight histopathological changes including cloudy swelling of the parenchymal cells of the liver and cloudy swelling of the tubular epithelium of the kidney. The significance of these effects was not explained.

In man and in animals, ethylbenzene is an irritant of mucous membranes. It is this response which forms the basis for the current Threshold Limit Value (TLV). The U.S. EPA recommended carcinogenicity testing for ethylbenzene in 1976, but test results are not yet available. Similarly, no data exist for mutagenicity and teratogenicity of ethylbenzene. The potential adverse human health effects following exposure to ethylbenzene were stated (40 FR 1910.1034) to be:

1) kidney disease,

Ethylbenzene is not nephrotoxic. Concern is expressed because the kidney is the primary route of excretion of ethylbenzene and its metabolites.

2) liver disease,

Ethylbenzene is not hepatotoxic. Since ethylbenzene is metabolized by the liver, concern is expressed for this tissue.

3) chronic respiratory disease.

Exacerbation of pulmonary pathology might occur following exposure to ethylbenzene. Individuals with impaired pulmonary function might be at risk.

4) skin disease,

Ethylbenzene is a defatting agent and may cause dermatitis following prolonged exposure. Individuals with pre-existing skin problems may be more sensitive to ethylbenzene (U.S. EPA, 1980 c)

Rationale for Denial of Dow Petition

As a result of its review the EPA concludes that available data support the listing of phenol, 2,4-dicholorophenol, 2,4,5-trichlorophenol, pentachlorophenol, and ethylbenzene.

1. Phenol warrants listing because of the combined effects of high toxicity to aquatic life, its ability to promote the carcinogenicity of weak carcinogens, its widespread occurrence in industrial effluents and the aquatic ecosystem, and its potential to persist under certain environmental conditions.

2. Pentachlorophenol warrants listing because it is highly toxic to aquatic organisms, bioaccumulates, and is

widespread in industrial effluents and in the aquatic ecosystem.

3. 2,4-Dichlorophenol and 2,4,5-trichlorophenol warrant listing because they are highly toxic to aquatic organisms, they promote the carcinogenicity of other chemicals and they are discharged in point source effluents.

4. Ethylbenzene warrants listing because it occurs in industrial effluents and the aquatic environment and it bioaccumulates in man and may bind to sediment. The current toxicity data base for ethylbenzene is sufficient to warrant delisting since there is no assurance that adequate control of this chemical will not be compromised (Cong. Reg. daily ad. S. 19649).

Dated: December 24, 1980.

Douglas M. Costle,

Administrator.

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[WH-FRL 1701–7]

Proposed Denial of a Dow Chemical Company Petition To Remove Monochlorophenyl Phenyl Ether From the Clean Water Act Section 307(a)(1) Toxic Pollutant List

AGENCY: Environmental Protection Agency.

ACTION: Proposed denial of a petition to remove monochlorophenyl phenyl ether (ClDPO) from the class of compounds, haloethers, on the toxic pollutant list under section 307(a)(1) of the Clean Water Act, as amended, 33 U.S.C. 1317(a).

SUMMARY: This action notices receipt of a petition from Dow Chemical, USA, to remove monochlorophenyl phenyl ether, hereafter referred to as ClDPO, from the toxic pollutant list. It addresses the petition's contentions, discusses data submitted by Dow in support of the petition, reviews additional information available to the Agency regarding this chemical, and affirms that ClDPO be retained on the toxic pollutant list. Dischargers of chemicals listed under section 307(a) of the Clean Air Act are subject to the most stringent technological controls representing Best Available Technology Economically Achievable (BAT). BAT is anticipated to be more costly than technologies resulting from waivers from BAT (granted to industrial dischargers of nonconventional pollutants) or technologies representing BCT (control technology for conventional pollutants. The denial of this petition precludes reclassification of the chemcial thereby eliminating the possibility for industrial use of alternative technologies and resultant lessening of economic impact.

DATES: Public comments on this proposed denial must be submitted on or before March 9, 1981, 1981, to Mr. Joseph A. Krivak at the address listed below.

ADDRESS: Criteria and Standards Division (WH–585), Office of Water Regulations & Standards, Environmental Protection Agency, 401 M Street, S.W., Washington, D.C. 20460.

FOR FURTHER INFORMATION CONTACT: Mr. Joseph A. Krivak, Director (202–755–0100).

SUPPLEMENTARY INFORMATION: Section 307(a)(1) of the Clean Water Act requires EPA to publish a list of toxic

pollutants and also authorizes the Administrator to add or remove substances from this list. These determinations are to be based upon the following statutory standard:

> The Administrator, in publishing any revised list, including the addition or removal of any pollutant from such list, shall take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms in any waters, the importance of the affected organisms, and the nature and extent of the effect of the toxic pollutant on such organisms.

As the Agency interprets this standards, the authority granted is essentially discretionary. That is, the Administrator is to consider each of the enumerated statutory factors, but has leeway to weigh the importance of each factor, and of data submitted pursuant to each factor. In making this balancing determination, special attention will be paid to potential impact on public health and the aquatic food chain.

On March 27, 1979 (44 FR 18279), EPA published a notice identifying data deemed relevant in procesing section 307 petitions. The purpose of the notice was to provide guidance to persons petitioning EPA for a change in the toxic pollutant list.

The purpose of the section 307(a) list of toxic pollutants is to focus attention on the potential significant environmental harm which might result if such pollutants are discharged to the aquatic environment and to control such harmful discharges through the development and implementation of appropriate effluent limitations. Toxic pollutants are subject to BAT (as are non-conventional pollutants) and (unlike non-conventional pollutants) are not eligible for waivers on water quality or economic grounds.

Background

On June 7, 1978, the Agency received a petition from the Dow Chemical Company requesting the removal of aromatic haloethers, found in section 307(a) under the general class "haloethers", from the toxic pollutant list. The subject compound, ClDPO, is an aromatic haloether. The Agency reviewed data supplied by Dow to support their petition and reviewed other available data as well. Some of the data pertained to ClDPO. From this review, the Agency concluded that the entire group of aromatic haloethers, including ClDPO, should be retained on the list. Accordingly, we published a proposed denial of the Dow petition in 44 FR 18279, March 27, 1979. After consideration of public comments, the Agency published a final denial of the petition in 44 FR 59948, October 17, 1979.

On February 22, 1980, the Agency received another petition from Dow requesting that ClDPO alone be removed from the toxic pollutant list. The Agency has tentatively determined to deny the petition. ClDPO should be retained on the toxic pollutant list because data indicate it is toxic to aquatic organisms and mammals, may pose a human health threat, is present in effluent discharges and fish tissue, and bioconcentrates to a significant degree in aquatic organisms. EPA's detailed responses to Dow's specific contentions in their petition are set forth below.

(1) Dow contends that according to the EPA document, *The Process of Selection and Prioritization of Toxic Pollutants in Point Source Water Effluent Discharges,* ClDPO has not been found in municipal or industrial wastewater effluent discharges.

EPA Response

EPA, in fact, has identified ClDPO in both aquatic organisms and in industrial effluents. Thus, EPA has identified ClDPO in aquatic biota of the Pacific Northwest. Some observations are: 0.05 mg/kg of ClDPO was found in lake trout tissue from Galbraith Lake near the Antigun River in Alaska; 0.10 mg/kg of ClDPO was found in fish tissue taken from the Kenai River near Soldotna in Alaska (*1*).

ClDPO likewise has been identified in the untreated effluent of a paint manufacturer in New Jersey at a level of 266 μg/1 (*8*).

ClDPO has also been identified in eight different samples of the effluent of a company in Kansas at levels of about 20μg/1. The discharge area drains into Cowskin Creek and then into the Arkansas River (*8*). Exposure of affected organisms to ClDPO, therefore, is not only possible but in fact is occurring.

(2) Dow states: "ClDPO has only one known commercial use i.e., in a mixture for a dielectric fluid. In this use, the fluid is hermetically sealed within capacitors. Human exposure is minimal during production and processing for use, and when disposed of by simple incineration or in an approved landfill, contamination of the environment is completely avoided."

EPA Response

We disagree with Dow's conclusion. It may be true that at the present time ClDPO is only used as a component of dielectric fluid; however, this does not preclude it from being used otherwise in the future as new applications develop. Moreover, the fact that ClDPO is hermetically sealed within capacitors may not preclude evironmental hazard. PCB's in their applications are also hermetically sealed and yet are now found in many waters throughout the world. In any case, other exposure pathways for ClDPO are available. ClDPO is initially prepared as a separate chemical (*2*). It can be potentially stored or shipped as a separate chemical and therefore, there is always the possibility of an accidental spill or discharge. The fact that ClDPO has been found in aquatic biota and in industrial effluents demonstrates that the chemical has found its way into the aquatic environment.

Dow's claim that this chemical may safely be disposed of through simple incineration or in approved landfills may or may not be true. The claim is unsubstantiated and is entirely irrelevant to a listing determination under section 307(a). Waste disposal practices are, of course subject, to Federal and State regulations under the Resource Conservation and Recovery Act of 1976 (RCRA).

(3) Dow states: "ClDPO is a relatively low-volume production chemical. In 1977 and 1978, production was well under 300 thousand pounds. Also, the market for ClDPO is not expected to exceed one million pounds annually. Production quantities like these are not in the same category as most materials on the toxic pollutant list."

EPA Response

We believe that currently there is a sufficient volume of ClDPO being produced and a potential exposure to affected organisms that exists to warrant concern for environmental hazard. Dow did not furnish production and/or use figures for 1979, but did indicate that production by Dow would not exceed one million pounds in the near future.

While this is Dow's best estimate for future production, this does not include potential for imports and other manufacturers producing ClDPO. Since ClDPO has not been widely used until now, it is possible that limited monitoring and sampling programs have simply not yet detected its presence in more than two or three regions of the United States. Since Dow states that future production will be increased at least threefold for ClDPO, the potential for increased future discharges exists.

(4) Dow states: "Environmental and mammalian toxicity data and the expected use-exposure pattern for ClDPO indicate a very low level of environmental and human hazard."

EPA Response

Dow has failed to substantiate this assertion. Dow's own data submitted in the previous petition for the removal of all aromatic haloethers demonstrated significant toxicity for ClDPO.

With respect to aquatic toxicity, acute data on freshwater organisms commonly used in toxicity testing show significant toxicity for ClDPO. The static 96-hour LC_{50} value for ClDPO on fathead minnows was 1.75 mg/1 (*3*). In a flow-through test on the same species, the threshold LC_{50} value for fathead minnows was 0.090 mg/1 (*3*). The static 48-hour LC_{50} value for *Daphnia magna* was 0.39 mg/1 (*3*). Chronic toxicity test data for aquatic organisms are not available.

Dow attempts to counter these data by postulating an antagonistic effect on fish toxicity between ClDPO and the butylated derivatives of ClDPO. Dow claims that the presence of these butylated materials as a mixture in the commercial capacitor fluid reduces the acute aquatic life toxicity of ClDPO by 1½ orders of magnitude. While this may be true, EPA under section 307(a) does not evaluate mixtures of compounds. The properties of ClDPO must stand or fall on their own merits. Furthermore, since the compound is prepared alone before being formulated into the capacitor fluid mixture, ClDPO has the potential to be stored and shipped alone and, therefore, be introduced into the aquatic environment as an individual compound.

With regard to mammalian toxicity, Dow introduced data from a sub-chronic study of rats conducted at its toxicology laboratory (*4, 10*). The rats were fed doses of 0, 5, 15, 45 and 90 mg/kg body weight/day of a mixture of 20% ClDPO and other mono-, di-, and tri-butyl derivatives of ClDPO (the dielectric fluid) for as long as 156 days (*4*). The major portion of the toxicological observations were made during the first 91 days (*10*). Dow stated that a number of parameters were unaffected including demeanor, clinical chemistry determinations, urinalyses, hematology, urinary excretion of coproporphyrin, uroporphyrin, creatinine and delta-aminolevulinic acid, and the weights of the brain, heart and testes at the time of sacrifice (*4*).

The dielectric fluid affected body weight, food consumption, and pathology (*10*). Body weight and food consumption of the treated rats were lower than those parameters of the controls. The pathology and organ weight data suggested that the liver and kidney were adversely affected and were toxicologically the most sensitive organs responding to the dielectric fluid. Even though Dow stated that these effects were "mild in degree, reversible in nature and none progressed in severity or nature throughout the rest of the study" (*4*), definite mammalian toxicity of this dielectric fluid was demonstrated by this study. It must also be remembered that this test was conducted with a mixture containing only 20% ClDPO along with other butylated derivatives of ClDPO. Therefore the results of these tests are probably not indicative of the effects of ClDPO alone.

In a further toxicity study conducted by Dow on rats' and rabbits' embryonal and fetal development, the 20% mixture was also used. Dow cited data that indicated a decrease in maternal weight gain and an increase in liver weights in the rat (*4*). EPA is not considering this positive toxic response to be relevant to our analysis because it also pertains to the mixture.

A single-dose oral feeding study conducted on guinea pigs demonstrated the acute toxicity of ClDPO alone toward non-human mammals (*11*). Four-day and thirty-day lethal doses were 700 mg/kg body weight and 600 mg/kg body weight, respectively. Since EPA's policy is that toxic responses to a chemical in non-human mammals can indicate a potential hazard for that chemical to human health (44 FR 15976, March 15, 1979), we regard this positive response as indicative of a possible adverse effect on humans.

Another such indication is the Occupational Safety and Health Administration's (OSHA) occupational air standard for a number of aromatic haloethers, including ClDPO (*9*) (see 29 CFR 1910.1000 Table Z1). Although this standard reflects dangers posed by airborne exposure, we think it nevertheless reflects a regulatory recognition of potential toxicity of ClDPO to humans.

(5) Dow states: "Although no long-term carcinogenicity tests have been performed, the Ames mutagenicity test on microbes was *negative* for the 20% ClDPO mixture, *using* both *activated* and *non-activated* test systems." (emphasis original)

EPA Response

A compound need not be genetically active to be listed under section 307(a), other toxic effects alone being sufficient under the statutory standard. As indicated above, the Agency regards the aquatic life and mammalian toxicity of ClDPO as sufficient to justify its inclusion on the list.

Further, Dow's data by no means show conclusively that ClDPO is not genetically active. First, very limited data were forwarded with the petition and the testing protocol itself was not submitted. Second, Dow did not submit any Ames test data for pure ClDPO or any data on mammalian cell mutagencity testing for pure ClDPO or the 20% mixture. In any case, EPA has found that the Ames test is not the ultimate test of the mutagenicity or potential carcinogenicity of a chemical. Approximately ten percent of carcinogens tested (18/175) were non-mutagenic in the Ames test. Several chlorinated hydrocarbons among the 65 toxic pollutants tested negative in the Ames test and positive in full carcinogenicity testing. Examples include carbon tetrachloride, 1,1-dichloro-2,2-bis(p-chlorophenyl)ethene (DDE), and dieldrin (*7*). While these three compounds tested negative in the Ames test, they exhibited potentials for mutagenic activity as shown by other mutagenicity screening tests. For instance, they have been shown to interact with the DNA of mammalian cells.

(6) Dow states: "Our data on environmental transport and fate show that ClDPO is rapidly degraded in the activated sludge test, in the simple BOD test, and the river die-away test. Any ClDPO accidentally entering the aquatic environment will dissipate rapidly by biodegradation and evaporation. Absorption and bioconcentration in fish are reversible in nature, and therefore do not play an important role in determining ClDPO's fate in the aquatic environment."

EPA Response

It is important to note that biodegradation is a function of the presence of organisms capable of metabolizing the pollutant, of temperature, of the presence of nutrients, and of the chemical characteristics of the pollutant. Furthermore, biodegradation does not necessarily mean that the environmental pollutional load is lessened. Some degradation products are persistent and may be more harmful than the original pollutant. Dow fails to address many of these variables in its environmental fate discussion. Thus in the test submitted by the petitioner, no degradation intermediates were identified or biological properties assessed. Therefore, the extent of molecular degradation and potential toxicity of intermediate chemical species are not known for ClDPO.

Dow also indicates that the rate of formation of $14CO_2$ from ring-labeled ClDPO was mesured in activated sludge and compared with the rate of $14CO_2$

formation from a ring-labeled PCB isomer. ClDPO was found to degrade 1400 times faster than the PCB isomer (*3*). However, this comparison is not meaningful and is not a definitive method for determining relief of environmental pollution load. PCBs are extremely stable compounds in the aquatic environment and so the practice of comparing ClDPO's biodegradation rate to that of a PCB isomer is questionable for showing pollution load relief.

EPA disputes Dow's assertion that bioconcentration does not play an important role in determining the fate of ClDPO in the aquatic environment. Although the bioconcentration factor (BCF) of 736 for ClDPO in trout muscle cited in the petition (*5*) is lower than the BCF of 9,550 for a PCB isomer (*6*) the BCF of 736 for ClDPO is still significant. Moreover, data on a single tissue type from one aquatic vertebrates species is inadequate to demonstrate low bioconcentration potential. Bioconcentration data must be obtained in various tissues from several freshwater and marine species before a determination of insignificant bioconcentration potential can be made.

(7) Dow states: "The sediment-binding data shown in table 2 of the petition (bound to suspended solids—sediment/water ratio—4:1) represent the ratio of chemical bound to sludge to the concentration of chemical in the supernatant water from centrifuged sludge. Activated sludge simulates some aquatic sediments. Sediment binding ratios, like the 4:1 ratio for C1DOP, indicate such compounds will be bound to sediments reducing the concentrations that will remain in water."

EPA Response

EPA agrees that the ability of ClDPO to bind to sediments should reduce the concentration of ClDPO that would remain in the water column. However, ClDPO's association with sediments suggests a potential exposure problem for benthic organisms and bottom feeders. ClDPO, therefore, would not necessarily be removed from the aquatic food chain.

(8) Dow states: "Clearly ClDPO does not fit the definition of a toxic pollutant as defined by section 502(13) of the Clean Water Act. There has been no known discharge and, on the basis of the information available to the Agency, there has been no interference with water quality.

Finally, other sections of the Clean Water Act Amendments provide the Agency with authority to regulate chemicals like ClDPO when and if they become a health or environmental hazard. We do not believe that ClDPO poses such a hazard."

EPA Response

ClDPO has been detected in industrial effluents (*8*) and fish tissues (*1*). Therefore, actual exposure of ClDPO to the aquatic community has been demonstrated and potential exposure of ClDPO to humans has been suggested. In addition, acute fish toxicity (*3*) and moderate mammalian toxicity (*11*) have been demonstrated. These are some of the criteria used for placing or keeping a compound as a toxic pollutant under section 307(a).

Summary

For the reasons stated above, EPA has concluded that Dow has not presented sufficient evidence in its petition for the removal of ClDPO from the toxic pollutant list. EPA, therefore, proposes to deny the petition for removal of ClDPO from the toxic pollutant list based on the evidence presented herein.

Dated: December 24, 1980.

Douglas M. Costle,

Administrator.

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[WH–FRL 1701–8]

Petition To Remove Chlorodifluoromethane From the List of Toxic Pollutants Under Section 307(a)(1) of the Clean Water Act

AGENCY: United States Environmental Protection Agency (EPA).

ACTION: Request for public comment on a petition from Vulcan Materials Company to delete chlorodifluoromethane (R–22) from the list of toxic pollutants under Section 307(a)(1) of the Clean Water Act.

SUMMARY: This action notices receipt of and requests comments on a petition (and supporting data) from Vulcan Materials Company (Vulcan) to remove the refrigerant, chlorodifluoromethane (R–22) from the Section 307(a)(1) toxic pollutant list. The EPA is also requesting additional information on chlorodifluoromethane relating to its toxicity, persistence (including its mobility and degradability in water), bioconcentration, bioaccumulation, or biomagnification and octanol/water partition coefficent. Information is also requested on the extent of point source discharges, qualitative or quantitative determinations in industrial and municipal wastewater effluents, ambient water, benthic sediments, fish and other aquatic life and any other data relating to the potential for human, aquatic or wildlife exposure. EPA will evaluate all data and public comments before deciding on the listing status of R–22. The decision will be published in the **Federal Register** if the petition is denied or as a Final Rule if the petition is accepted.

DATES: Public comments on the petition and additional information will be received until March 9, 1981.

ADDRESS: Send comments to: Jacqueline V. Carr, Criteria and Standards Division (WH–585), Office of Water Regulations and Standards, U.S. Environmental Protection Agency, 401 M Street, SW, Washington, D.C. 20460.

FOR FURTHER INFORMATION CONTACT: Jacqueline V. Carr, Telephone (202) 245–3036.

SUPPLEMENTARY INFORMATION: On September 9, 1980, Vulcan Materials Company petitioned the EPA to remove chlorodifluoromethane (R–22) from the toxic pollutant list. Vulcan proposed the

removal of this chemical from the list of toxic pollutants under Section 307(a)(1) of the Clean Water Act based on an assertion that R–22 is similar in its physical and toxicological properties to the chlorofluorocarbons, dichlorodifluoromethane (F–12; R–12) and trichlorofluoromethane (F–11; R–11) which the EPA proposed to delist in 45 FR 46103 on July 9, 1980. Vulcan concluded that R–22 does not present a hazard to drinking water supplies or to the aquatic environment.

Vulcan Materials Company supplied the following information to support the petition.

Chlorodifluoromethane (R–22)

(1) This refrigerant gas has very similar physical and toxicological properties when compared to trichlorofluoromethane (R–11) and dichlorofluoromethane (R–12). R–22 boils at −41.4° F (−42.5° C). The solubility of water in R–22 is 1.2×10^6 ug/l and the heat of vaporization is 5,213 cal/g mole[1] as compared to 3,996 cal/g mole and 8,363 cal/g mole for R–11 and R–12 respectively. Additional physical constants may be found in the attachment[2] (Table 1).

(2) NIOSH has listed similar aquatic toxicity values for R–22 and R–12, i.e., TLm96>1,000.[3] Inhalation exposure to rats suggests that R–22 may be less toxic than R–11 with the LCLo for R–22 being 25 pph/4 hours as compared to 10 pph/20 minutes for R–11.

[1] *Handbook of Chemistry and Physics*, 53rd Ed., CRC Publishers.

[2] *Fluorocarbons and the Environment*, National Science Foundation, NSF 75–403.

[3] *The Registry of Toxic Effect of Chemical Substances*, NIOSH, 1978.

Table VI–1.—*Physical Properties of Selected Fluorocarbons: Methane Series (After Shepherd, 1961)*

Property	F–11	F–12	F–21	F–22	F–30
Chemical formula	CCl_3F	CCl_2F_2	$CHCl_2F$	$CHClF_2$	CH_2Cl_2
Molecular weight	137.4	120.9	102.9	86.5	84.9
Boiling point, °F	74.8	−21.6	48.1	−41.4	105.2
Freezing point, °F	−168	−252	−211	−256	−142
Vapor pressure, psig:					
At 70°F	[1] 13.4	70.2	8.4	122.5	[1]7.1
At 130°F	24.3	181.0	50.5	300	9.0
Liquid density, g/cm$_3$:					
At 70°F	1.485	1.325	1.323	1.209	1.325
At 130°F	1.403	1.191	1.193	1.064	
Vapor density at boiling point, g/1	5.86	6.26	4.57	4.83	3.30
Heat of vaporization at boiling point, Btu/lb	78.31	71.94	104.2	100.7	141.7
Liquid viscosity, centipoise:					
At 70°F	0.439	0.252	0.351	0.238	[2]0.441
At 130°F	0.336	0.227	0.286	0.211	
Liquid thermal conductivity at 70°F, (Btu)(ft)/(ft$_2$)(hr)(°F)	0.063	0.051	0.072	0.063	[3]0.059
Surface tension at 77°F, dynes/cm	19	9	19	9	[3]28
Solubility of water at 70°F, wt pct	0.009	0.008	0.13	0.12	[3]0.17
Flammable limits, vol pct in air	None	None	None	None	None
Toxicity: Underwriters' Laboratories, Inc. rating system	5A	6	[4](5)	5A	4–5

[1] In pounds per square inch absolute.
[2] At 68°F.
[3] At 77°F.
[4] Much less than 4, slightly more than 5.

Summary of Additional Available Data

1. Production and use

Five manufacturers in the U.S. operate 14 plants which produce chlorodifluoromethane. Production of R–22 in 1979 exceeded 200 million pounds. R–22 is used primarly as a refrigerant and has minor uses as a polymer intermediate, propellant and solvent (CEQ, 1975; personal communication, Vulcan Materials Company).

2. Persistence and Degradability

The hydrolysis rate of R–22 is reported to be less than 0.01 grams per liter of water per year (CEQ, 1975). The rate of hydrolysis is proportional to the concentration in solution and the hydroxyl ion concentration (Dupont, 1974). This rate is considered too low to be a significant aquatic fate process.

There are no data for other fate processes, although volatilization of R–22 is considered the dominant fate process based on the vapor pressure (10 atm at 24°C) and water solubility (3.0×10^6 μg/l @ 25°C and 1 atm.) of the compound (Dupont, 1974; Handbook of Chemistry and Physics, 1976).

There are no data which indicate that monitoring for R–22 has been done in industrial effluents, wastewater treatment streams, surface waters, fish or sediments.

Toxicity

A threshold limit value of 1000 ppm (35000mg/m^3) has been established by the American Conference of Governmental Industrial Hygienists (ACGIH) to protest workers from adverse effects of inhaling R–22 (Dupont, 1974). Human deaths associated with fluorocarbon propellant inhalation are attributed to cardiac arrhythmia (U.S. EPA, 1974). Belej et.al. (1974) exposed monkeys to inhalatory concentrations of R–22 of 10 and 20 percent. No arrythmia or tachychardia were observed but monkeys showed decreased myocardial contractability with a fall in aortic blood pressure. R–22 was also shown to sensitize the hearts of mice to epinephrine (Aviado and Belej, 1974).

R–22 is weakly mutagenic in *Salmonella typhmurium* strains TA 1535 and TA 100. Bacteria exposed for 24 hours to R–22 concentrations of 1, 33, and 50 percent mutated from a dependence on histidine to histidine independence in greater numbers than untreated bacteria. The presence of a rat liver microsomal activating system was not required for mutagenic activity (Longstaff and McGregor, 1978). The authors cautioned against the use of these data to assess risks to man until conventional animal studies are completed.

Haskell Laboratory (Dupont), 1977 conducted embryotoxicity and teratogenicity studies in rates exposed to chlorodifluoromethane by inhalation. Two studies were conducted, the first using female rats exposed to 0.1 and 1.0 percent (vol/vol in air) R–22, the second using females exposed to 0.05, 0.10 and 2.00 percent (vol/vol in air) R–22.

Gross external examination of all fetuses showed a few fetuses with minute hematomas and petechial hemorrhages in several litters in all control and test groups. One fetus in a litter of 3 in the high level group of the second study experienced anophthalmia (absent eye) in both eyes, lack or organization of the central nervous system and ossification. No major skeletal abnormalities were detected in either study. Several soft tissue malformations were observed in fetuses in both groups. Abnormalities observed were microphthalmia (small eye) and anophthalmia. Cleft palate was observed in one fetus exposed to 0.1 percent R–22. Subcutaneous edema (anasarca) was detected in two fetuses (0.05 percent and 2.00 percent R–22).

Under the conditions of the two studies, chlorodifluoromethane is a

weak teratogen. The incidence of abnormalities was not dose related but statistically significant when compared to historical control litters at Haskell Laboratories and Charles River Laboratories.

Studies of chronic inhalatory toxicity in rats and mice indicate that R–22 exposures of 0.198 percent given 6 hours/day for 300 days produce no toxic effects (U.S. EPA, 1974).

A study of the acute inhalatory toxicity of R–22 in laboratory animals indicates that as a concentration of 10% R–22 produced tremors in guinea pigs at 2 hours; 40% R–22 produced anesthesia in dogs in less then 90 minutes and concentration of 70% and 40% were approximate lethal concentrations (ALC—minimum concentration causing death over a given exposure period) to dogs (less than 90 min) and mice (2 hr), respectively (U.S. EPA, 1974).

There are practically no data on the acute and chronic toxicity of R–22 in aquatic organisms. NIOSH(1977) lists an aquatic toxicity rating (TLm 96) over 1000 ppm for an unspecified species.

Request for Additional Information

In revising the toxic pollutant list, the Clean Water Act directs the Administrator to take into account the toxicity of the pollutant, its persistence, degradability, the usual or potential presence of the affected organisms, the importance of the organisms and the nature and extent of the effect of the pollutant on such organisms.

EPA requests additional information on the fate and toxicity of F–22 in the Aquatic environment for the assessment of these statutory factors. EPA requests information on chlorodifluoromethane relating to its toxicity to aquatic life, persistence (including its mobility and degradability in water), bioconcentration, bioaccumulation, or biomagnification and octanol/water partition coefficient. Information is also requested on the extent of point source discharges, quanitative or quantitiative determinations in effluents, ambient water, benthic sediments, fish and other aquatic life and other data relating to the potential for human, aquatic or wildlife exposure.

Date: December 24, 1980.

Douglas M. Costle,

Administrator.

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